# TRICHINOSIS, BOTULISM, STAPHYLOCOCCAL FOOD POISONING: PREVENTION AND CONTROL



by

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Food poisoning and its prevention is an international problem, and has been a basic problem for man since his inception. The writings of ancient man identify foods which were believed to cause food poisoning, as well as toxic plants which served as poisons.

The first "method" followed by man to control food poisoning was abstinence. Records show that primitive tribes, who had not developed written communications, established certain "taboos" to prevent food poisoning by preventing consumption. They also provided for specific treatment of certain foods to assure safety, and in many instances, to also assure decency.

The American Indian found that the poisonous effect of acorns could be alleviated by soaking the nuts in water and extracting the poisonous tannins. The heating of some foods prior to consumption also served to prevent food poisoning.

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Early attempts to preserve meat by smoking, drying, freezing, salting, pickling, or fermentation were undoubtedly accompanied by food poisoning outbreaks, until it was learned that certain procedures must be followed if food poisoning was to be minimized. Meyer(1) points out that during the 9th century sausage making was accompanied by food poisoning outbreaks, as evidenced by the order of the Byzantine Emperor Leo VI (886-912). He was the first to pass laws preventing the consumption of blood sausage because of its harmfulness to health.

Man's early support of meat inspection suggests that it was initiated following the association of illness with the consumption of meat from sick animals. Unfortunately, recognizing the cause of food poisoning does not eliminate it, as evidenced by the recent outbreaks of botulism. In retrospect, it is incredible how much was learned about the prevention of food poisoning and how much prevention was practiced when superstition, exploitation of ignorance, and miasmal theories dominated and distorted man's objective evaluation of the facts.

It is equally difficult to understand why man has been reluctant to comply with scientifically proven methods for eliminating food poisoning. No better example can be cited than that of trichinosis, a disease which is transmitted only by consuming infected meat.

TRICHINOSIS

The mysteries surrounding pork poisoning were dispelled in 1859 with Zenker's(2) demonstration of the role of Trichinella spiralis. Zenker turned his material over to Virchow, the father of modern pathology, and Leuckart, the most eminent parasitologist of his time. By 1863, (102 years ago), Virchow and Leuckart had fully developed the epidemiology and pathology, as well as a control method for this nematode.

By then it was scientifically apparent that trichinosis in man could be prevented by any one of the following methods: (1) abstinence from the consumption of all meat (vegetarian method), (2) abstinence from the consumption of meat from animals which are known to be infected (Hebrew and Islamic Method), (3) identification and elimination of meat which contains trichina (Virchow's method), (4) treatment of meat to assure destruction of trichina before consumption (American method), and (5) elimination of trichina from the diet of meat-producing animals (ideal method).

In the same year, 1863, Virchow traveled the length and breadth of Germany insisting upon the necessity for microscopic examination of each hog slaughtered for food.

In seeking to prevent trichinosis, it was self-evident that the disease would no longer be a problem for man if it could be eliminated from food animals. Virchow's microscopic control measures were necessitated by the raw-pork consumption habits of Germans and Middle Europeans generally. However, his measures also promoted the elimination of

trichina from swine.

Garbage-fed swine constitute the principal danger of trichinosis in the United States. Changes in swine husbandry and feeding practices, the reduction of home slaughter, and the freezing of locally-slaughtered pork have all contributed to the marked decline in the incidence of trichina in swine in this country.

The successful control of trichinosis in man, through microscopic examination of pork, is dependent upon the inspection of each hog slaughtered. In the regimented society which Virchow knew and with which he was concerned, microscopic examination of each hog and carnivorous game carcass was possible. Therefore, microscopic examination was eminently successful.

Among the French, where pork has always been thoroughly cooked, trichinosis has never been a significant public health problem. It is noteworthy, however, that trichinosis became a serious problem in the German Army of Occupation in Paris during World War II. Similarly, German prisoners-of-war in the U.S.A., who had habitually consumed raw pork with impunity in Germany, suffered from outbreaks of trichinosis when they did not follow the standard cooking and processing procedures for trichinosis control.

The soundness of Virchow's recommendations are substantiated by the foregoing. Further, more than 60 years have passed since there has been an outbreak in Germany of trichinosis in man traceable to inspected pork.

Opponents of trichinoscopic examination control argue that very light infections would be passed without recognition. This can be proved statistically. However, the German experience demonstrates that an infection rate of less than one trichina per gram does not present a significant public health hazard.

Following passage of the Federal Meat Inspection law in the U.S.A. in 1906, it was obvious to Federal authorities that microscopic examination of pork to control trichinosis in man could be successful only if every individual swine carcass was microscopically examined. Since only part of the pork supply was federally inspected, this practice would give consumers a false sense of security as to the safety of raw pork. It was feared that this false security would promote the consumption of uncertified raw pork, resulting in an increase of trichinosis in man.

The alternative for controlling trichinosis in man was to destroy all trichina in pork products that are customarily eaten without further cooking. The consumer also had to be educated to thoroughly cook other pork products.

The Meat Inspection Division, in cooperation with other agencies of the Department of Agriculture, developed thermal death times, freezing viability standards, and salt-concentration-time-temperature data covering the destruction of trichina in meat. These were incorporated in the meat inspection regulations, and have proven adequate to protect the public from trichinosis that might otherwise have been transmitted through processed pork products.

An intensive campaign employing veterinarians, educators, and public health officials was launched to educate consumers. This educational campaign, with its continued reiteration of the danger of eating improperly prepared pork, emphasized trichinosis to such an extent that many surveys of the prevalence of trichina infection in man were made in the early 1900's. These were based upon the examination of human muscles, almost all of which came from the bodies of elderly persons. Since any trichina infection leaves evidence to be revealed at time of death, an objective evaluation of the current status of trichinosis was not possible at that time.

These surveys showed the rate of trichina infections in man prior to the initiation of the Federal Meat Inspection program.

The average trichina rate was found to be approximately 17 percent for all surveys. This resulted in a widely quoted and generally accepted belief that one person in six in the United States has trichinosis. This was never true. The vast majority of positive human muscle samples contained so few trichina that awareness of the infection would not have been possible.

More recent surveys in the United States, in which the ages of the cadavers were recorded, show that eight cases of human trichina infestations occurred for each 1,000 years of life before 1910. Among those people born since 1910, however, the rate is only slightly more than one case per 1,000 years of human life. (3)

The United States Public Health Service's Morbidity and Mortality Reports for the last decade show an average of 250 clinical trichinosis cases with five deaths out of the 1,600,000 deaths reported annually. These figures have added significance when it is realized that 80 - 85 million swine are consumed each year in the United States.

Today, trichinosis in swine in the USA appears much less frequently than in 1900. Reliable statistics for the years 1890 - 1905 show that the incidence of trichinosis in farm-raised swine was 2.6 percent, and 12-20 percent in garbage-fed swine. A recent survey,(4) made from April 1961 through March 1965, of 21,417 swine examined by the artificial digestion technique using 45.4 grams of diaphragm muscle, showed the following rates of infection: (1) farm-raised butcher hogs, 0.12 percent; (2) farm-raised breeder hogs, 0.22 percent; and (3) garbage-fed hogs, 2.6 percent. Though garbage-fed hogs constitute only 1-1½ percent of the hogs produced in the United States, their importance from a trichinosis control standpoint is paramount. The intensity of infection in garbage-fed hogs, i.e., the number of trichina per gram, exceeded that of farm-raised hogs by more than 50 times. Only one farm-raised hog in 5,444 contained more than one trichina per gram, while an infection rate greater than one per gram was found in one out of 96 garbage-fed hogs.

The elimination of trichina from the diet of meat-producing animals is a problem which still exists, although it has been recognized for 102 years.

We might conclude that man is more willing to comply with procedures established by supposition and superstition than those proven to be practical by the scientific method.

No current discussion of this subject would be complete without recognition of the potential significance of the recently isolated strains of this parasite in Africa. Differences in virulence and pathogenicity for different species suggests that confinement to the areas it now invades should be of major importance. The apparently uniform characteristics of Arctic and north temperate zone strains have not created a problem in connection with established control methods in the northern hemisphere. The significance of the similarities between the current Italian and African strains needs careful evaluation. The use of thiabenzadole and other therapeutic agents for the treatment of the disease in man provides hope for the further alleviation of this affliction.

BOTULISM

The most spectacular, deadly, and widely-publicized food-poisoning is caused by Clostridium botulinum types A, B, $C_a$, $C_b$, D, E, and F. These are anaerobic, spore-forming bacteria which produce a neuroparalytic exotoxin in the food prior to consumption. The types are separated by the immunologically distinct toxin which each produces. The clinical disease which each type causes in man is so similar as to be indistinguishable.

The botulinic syndrome has been recognized as a specific clinical disease since the 18th century, when physicians in Southern Germany identified it as "die Würstvergiftung" (sausage-poisoning).

The Russians identified the botulinic syndrome which occurred in association with fish as icthyism. Awareness of this syndrome by the medical profession, generally, led to its recognition throughout the Western World.

In 1895, following an outbreak in Ellezelles, Belgium, three of 34 musicians who ate from a 10-day old salted ham died within one week, and 10 others almost died. All who ate from the ham showed symptoms of the neuroparalytic syndrome in proportion to the amount of meat consumed. Professor E. van Ermengem,(5) of the University of Ghent, isolated an anaerobic, spore-forming, toxin producing rod which he named Bacillus botulinus.

The next isolation of this organism was made by Landmann,(6) who investigated the first reported botulism case resulting from food other than meat or fish. This occurred in 1904 in Darmstadt, Germany, when 11 of 12 persons who ate a salad composed of preserved wax beans died. The survivor arrived late to the dinner, and his bean salad was heated with the other food prior to consumption, proving the heat lability of the toxin.

In 1910, Leuchs[7] worked with the van Ermengem and Landmann isolates and showed that the toxins were immunologically distinct, as the antitoxins would not protect one against the other. Though the cultures were soon lost, it is now generally agreed that van Ermengem's organism was a non-proteolytic type B, while Landmann's was type A.

Beginning in 1913, a series of outbreaks occurred in the United States causing high mortality. The deaths occurred principally in California, from home and commercially-canned non-acid fruits and vegetables. This resulted in a widespread investigation which led to the initiation of governmental controls.

The developing canning industry recognized that its future welfare depended upon its ability to prevent food poisoning. Thus, the industry gave full support to investigative, corrective, and preventive measures. Thermal death-time studies established heat processing schedules, which, when applied, have been 100 percent effective for commerical canners.

In 1921 and 1922, Meyer, Geiger, and Dubovsky,[8, 9, 10, 11, 12] reported on the widespread incidence of C. botulinum types A and B in the soils of the United States, Canada, Alaska, and much of Europe. Types A and B were the only recognized types at the time. In 1922, Bengston,[13] while studying wild-duck poisoning in alkiline lakes, isolated a third type from the larva of the green fly, Lucilia caesar. In the same year, Seddon[14] isolated a fourth type, with an immunologically distinct toxin, from cattle suffering from bulbar paralysis in Australia. These two types were later identified as C. botulinum type $C_a$ and $C_b$.

In 1927, Thieler and Robinson[15] isolated a fifth type of C. botulinum from cattle in South Africa. This type was later shown to have a specific toxin. It is identified as C. botulinum type D, and is the cause of "Lemziekte" of cattle. The geographical distribution of this organism appears to be restricted to the arid veldt of South Africa.

It was not until 1936 that C. botulinum type E was indentified by Gunnison, Cummings, and Meyer,[16] although it had been isolated from smoked fish by Hazen[17] in 1932. Type E, up to 1964, had been recovered from 84 human outbreaks in Japan, U.S.A., Canada, USSR, Denmark, Sweden, and Norway.[20] In almost all cases, it was associated with fish or marine mamals. It is known to be widely distributed in sea and lake mud, silt, water, and similar environs. It differs from the other types in its susceptibility to heat, its ability to grow and produce toxin at low temperatures, its ability to produce toxin in other than optimum anaerobic conditions, and its increased toxicity when treated with trypsin.

Type F was identified as a new type in 1961 by Dolman,[18] from a culture isolated by Møller and Scheibel[19] from homemade liver paste in Denmark. In most characteristics, except in the individuality of its toxin,type F resembles type A. This strain, according to Dolman,[20] appears to be recognized infrequently, not because the spores are heat sensitive, as is the case with type E, but because of its sparse distribution in nature.

Types A, B, and E have caused almost all of the botulism in man. The extremely high resistance to radiation of C. botulinum spores indicates that there is little hope for the widespread use of radiation alone to prevent botulism. The preventive measures for types A and B, which were recognized 40 years ago, are still the most effective today. They are: (1) heat processing of canned goods in accordance with the standards of the National Canners Association, (2) acid-preserved foods should have a pH no higher than 4.5, (3) refrigeration of perishable foods at temperatures below 8°C, and (4) hygienic practices to hold spore contamination to a minimum. The prevention of type E botulism presents a new control problem.

It is now generally recognized that vaccum packaging of smoked fish in the United States does not promote the growth and toxin production by the C. botulinum type E organism. Rather, vacuum packaging prevents the growth of the molds and other strict aerobic spoilage organisms which would cause the food to spoil in the absence of refrigeration. This extension of shelf-life permitted the growth and toxin production of the botulinum organism.

The Food and Drug Administration has established standards for the processing of smoked fish. These standards include heating to 82.2°C for 30 minutes, and the immediate freezing of the product after cooking and/or smoking.

The extensive research investigations being carried on in the United States and throughout the world will no doubt provide new methods of control. In the meantime, the consumer can be protected by careful sanitation, time-temperature, storage, and distribution controls, and the avoidance of practices designed to extend shelf-life without preventing C. botulinum growth. The food habits of the Indians of the Northwest, the Eskimo, and some of the Orientals, will need to be revised if botulism among these groups is to be avoided.

Therapy of the botulinum victim by the use of specific anti-toxin appears to have proved its value in those cases of moderate toxin intake which can be treated reasonably early. The demonstrated success of immunizing those working with botulinus organisms and toxins makes its use mandatory in the botulinum laboratory.

The published proceedings (21) of a symposium on botulism, sponsored by the Public Health Service, are recommended to those interested in a current and comprehensive coverage of this subject.

## STAPHYLOCOCCAL FOOD POISONING

Because of the ubiquity of staphylococci, its role in food poisoning was not recognized until 1913. Barber (22) identified the etiology of an explosive, violently nauseating, selectively occurring food poisoning as being due to the growth of a single strain of staphylococci. The food poisonings occurred in visitors to a Philippine farm who drank milk which had been held over night. This organism occurred regularly in the milk from one quarter of a cow's udder. Barber's report was overlooked until 1930, when Dack et al, (23) at the University of Chicago, isolated an enterotoxigenic staphylococcusfrom sponge cake and proved its toxigenicity on human volunteers. Dack also demonstrated the heat stability of the enterotoxin. Following Dack's (24) irrefutable evidence, it was possible to identify the causes of many carefully recorded food poisoning outbreaks, whose etiology had been undetermined or erroneously placed. Only man, monkeys, and cats react to the enterotoxin, and in varying degrees for each individual. Similarly, immunological response to the enterotoxin is widely variable. Coagulase negative staphylococci have never been shown to be enterotoxigenic, while only a small percentage of coagulase positive strains produce enterotoxin.

The cat test for enterotoxin is frequently non-specific because of its reaction to staphylococcal alpha and beta hemolysins.

In 1963, Casman (25) perfected his long-sought method for the serological identification of the presence of staphylococcal enterotoxin in meats. This initiated a new era for the identification and understanding of the staphylococcal enterotoxin problem. It had long been recognized that enterotoxin type A was the usual cause of food poisoning, and that enterotoxin type B occurred primarily when the normal intestinal microbial flora was destroyed by the use of antibiotics, allowing for the unrestricted proliferation of the antibiotic-resistant enterotoxingenic staphylococci.

The Meat Inspection Division, now a part of the Consumer and Marketing Service of the U. S. Department of Agriculture, has long been vitally concerned with the staphylococcal food-poisoning problem. In the mid 1930's, the meat industry developed a rapid cure, tenderizing process for hams. It was based upon the injection of the curing pickle, via the femoral artery, into all of the tissues. The injected ham was heated to a minimum of 137°F during the smoking process. Following the acceptance of this procedure by the Meat Inspection Division, a number of staphylococcal food-poisoning outbreaks occurred in association with these hams.

It is now recognized that the heat treatment killed the vegetative microbes. When chance contamination with an enterotoxigenic staphylococci occurred following heat treatment, the staphylococci would grow readily without the competition of other organisms. This did occur, resulting in an appreciable number of staphylococcal food poisoning cases due to such ham.

The meat industry had handled cured, unpasteurized hams without refrigeration or food poisoning for hundreds of years, and did not recognize the need for continuous refrigeration of pasteurized hams. The meat industry's own scientists, as well as those of the government, alerted the industry to the new problem created by the destruction of the normal microbial flora. The necessity for proper refrigeration throughout handling soon became well established, and such hams have not been a significant problem since.

No case has been identified in the United States of staphylococcal food poisoning, due to the development of staphylococcal enterotoxin in unpasteurized meat. It would seem logical that the thermal stable enterotoxin would survive the low temperature often used to cook meat, although this apparently has never happened. The Food and Drug Administration of the U. S. Department of Health, Education, and Welfare, and the Meat Inspection Division jointly investigated this problem. The Meat Inspection Division laboratories attempted to grow enterotoxigenic staphylococci on raw meat. This attempt failed when large numbers of enterotoxigenic staphylococci were inoculated into ground beef with minimal contamination of other organisms. However, inoculation of non-contaminated surfaces with low numbers of the same strains of organisms resulted in luxuriant growth and toxin production. The Food and Drug Administration perfected the technique for serological identification of the enterotoxin.

McCoy, of our laboratory, has continued these studies. Preliminary results indicate that enterotoxin formation does not necessarily accompany good staphylococcal growth, and that concomitant organisms may enhance or inhibit enterotoxin formation.

Data obtained from the growth of two enterotoxigenic strains of Staphylococcus aureus (196E and 265-1) grown in shake cultures of a ham slurry (1 part ham + 2 parts distilled water) in association with organisms representing 13 different genera are presented below. Staphylococcal growth, when grown in association with another organism, was equal to or better than that grown in pure culture, except when grown with Serratia marcescens. In the latter case there was a 10 fold difference between the "dual" culture and the control after 24 hours at 35°C. Two organisms were inhibited by the staphylococci. Bacillus megaterium and Brevibacterium linens failed to grow in association with the staphylococci. Bacillus cereus seemed to enhance enterotoxin production by strain 196E. Readily detectable levels of enterotoxin were formed by strain 265-1 after 24 hours when grown with Alcaligenes faecalis, Alc. viscolactis, Bacillus cereus, Brevibacterium acetylicum, and Klebsiella pneumoniae, while none was detected when grown with Aerobacter aerogenes, E. coli, Micrococcus freudenreichii, Proteus mirabilis, Proteus vulgaris, Pseudomonus sp., Salmonella enteritidis, Sarcina subflora, and Serratia marcescens.

Inhibition of enterotoxin formation occurred even though in many cases there were one or two log increases in the number of staphylococci.

These data represent only some of preliminary work, more detailed and complete work will be published in the near future.

It now appears that humans are more likely to be the source of enterotoxigenic staphylococci than are lower animals. In a recent survey, Casman[26] reported that 50 staphylococci isolated from bovine mastitis failed to reveal a single enterotoxigenic strain, while staphylococci isolated from the posterior nares and skin infection of man frequently contained enterotoxigenic strains. Immunologically distinct types of staphylococcal enterotoxin, other than types A and B, have been recognized but not yet classified. This complicates the problem of identification of the enterotoxigenic staphylococcus.

In view of current hygienic practice, it is unrealistic to expect that human food will be handled without contamination with staphylococci, some of which will be enterotoxigenic. Therefore, the significance of the presence of staphylococci in a food must be evaluated with these considerations: (1) does it contain enterotoxigenic staphylococci, (2) are they present in sufficient numbers to indicate that significant enterotoxin has been produced, and (3) do massive numbers of dead cocci in suspected food require a determination for enterotoxin? It is generally believed that enterotoxigenic staphylococci must be present in a food in excess of one million per gram before sufficient enterotoxin is produced to induce food poisoning.

Staphylococcal food poisoning can be prevented by the following: (1) maintenance of the normal microbial flora in cured and fresh meats, (2) pasteurization followed by continuous refrigeration, and (3) pasteurization and prevention of re-contamination. Relying solely upon the third method can be a dangerous practice, because of botulism and the possible inapparent growth of unrecognized staphylococcus or other pathogen contamination.

In a discussion of microbial food poisoning, it is appropriate to evaluate the significance of bacterial numbers. Bacterial numbers are of tremendous significance in foods. Their enumeration and identification in meat serves many useful purposes, such as (1) identification of pathogenic organisms brings recognition of potential danger, (2) enumeration and identification of types indicate contamination with filth or gross contamination, (3) enumeration of spoilage organisms allows for estimation of expected shelf-life, (4) regular on-the-line microbial evaluations often identify a violation of proper sanitary practice, so that corrective measures can be taken, and (5) determination that desirable curing organisms predominate. Many other specific microbial evaluations for specific problems are in use, and others can be devised to serve special problems.

However, in the evaluation of the fitness or unfitness of meat for food, based upon bacterial numbers of spoilage organisms, the Meat Inspection Division has been unable to establish numbers which are meaningful in view of the great variability of numbers associated with organoleptic change.

The most precise evaluation of unfitness of meat for food due to decomposition is still organoleptic evaluation by an experienced person.

CONCLUSION

Analysis of reports of food poisonings within the United States shows that, in almost every case, gross violations of recognized hygienic practices usually near the serving time have been demonstrated. Hope for the complete elimination of food poisoning is dependent upon compliance with established hygienic practices by the public at large. In the meantime, education of the public, continual vigilance by public health authorities, and research into the causes of fish poisonings, mycotoxins, and probably other unidentified problems, require international awareness and support.

When one recognizes the extent of the contamination of food with pathogenic microbes, one wonders at man's survival. Evaluation of this circumstance clearly shows that non-pathogenic microbes, which are normally present in the food, either overgrow the pathogens, prevent the production of toxins, or spoil the food so that it is not consumed. In any event, man's greatest protection from microbial food poisoning has been the so-called spoilage organisms.

Practices designed to, or which incidentally, eliminate or control spoilage organisms unaccompanied by effective controls of pathogenic organisms have created most microbial food poisonings.

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